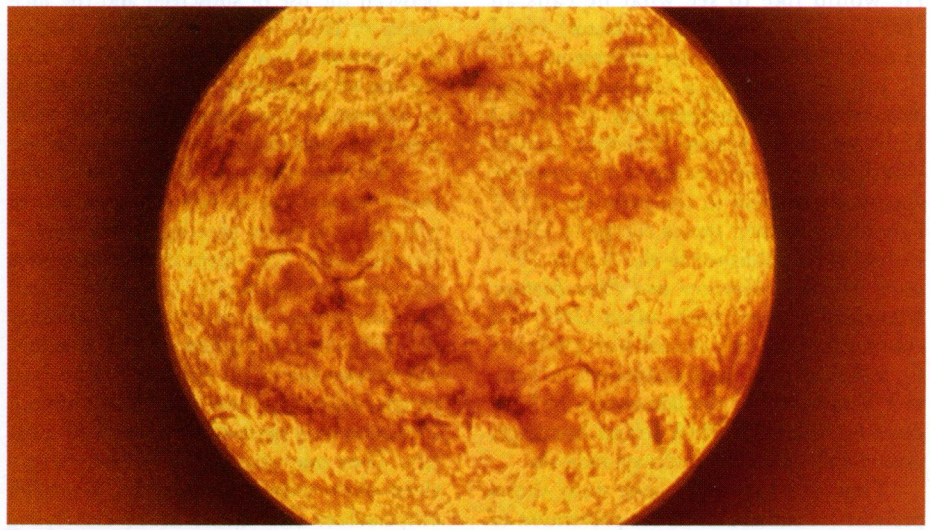
heightened by the tenant's belief that he is powerless, by dint of finances, to control his circumstances. Stansfeld explains, "It is hypothesized that exposure to environmental factors such as noise may be part of the explanation for differences in coronary heart disease by social status, and that, in turn, psychosocial factors such as perceptions of control may explain how social status influences primary risk factors for coronary heart disease such as blood pressure." But, Stansfeld continues, scientists are still uncertain of how and even whether long-term health effects are caused by noise exposures. "It is well known that sudden noise may cause short-term responses, such as raised heart rate," he says, "but longer-term changes that might affect health are important but unknown."

A frequent criticism among the report's reviews is the lack of consistent, adequate measurement of noise exposures. Furthermore, says Stansfeld, while it is possible to establish a reliable measure of annoyance as a subjective response to noise, it is much more difficult to establish whether noise is causing changes in bodily physiology. Bernard Berry, head of noise standards at the National Physical Laboratory in Teddington, the United Kingdom, and president of the U.K. Institute of Acoustics in St. Albans, explains, "All too often, the measurement and description of the physical exposure is regarded as of secondary importance, and yet it is one of the key components in enabling us to relate and compare different research findings. This points to the need for researchers to make use of internationally standardized measures, such as ISO 1996 [an internationally agreed-upon set of methods and units for measuring environmental noise], but also to retain sufficient flexibility in the measurements of noise exposure to allow us to investigate the possible importance of other measures."

The report concludes that, despite the uncertainties remaining about noise's nonauditory effects, there are sufficient data to warrant further study. The report recommends longitudinal studies that, while expensive in the short run, may very well yield cost-saving insights into how and why noise affects human health.

Studies Shed Light on Sunscreen Efficacy

The relationship between use of sunscreen and prevention of skin cancer remains unclear despite intriguing results of several studies presented on 17 February 1998 at the American Association for the Advancement of Science annual meeting in Philadelphia, Pennsylvania. According to these studies, consumers can't be certain which sunscreen, if any, will lower their risk for any of the three types of skin cancers. Two preliminary studies even suggest that using sunscreen may increase cancer risk.



False sense of security? New information shows that use of sunscreen may not be enough to protect against skin cancer, and people's dependence on them could actually lead to increased risk.

Sunscreens are formulated to protect against sunburn, and, though a prophylactic benefit has long been assumed by both the public and academia, there is little evidence that preventing sunburn in human skin prevents skin cancer. It is well-established that 90% of skin cancers are caused by exposure to light, but the causal mechanisms for basal cell carcinoma (BCC) and squamous cell carcinoma (SCC)—both known as nonmelanoma cancers—and melanoma are only now being probed. In the United States, according to Marianne Berwick, an associate attending epidemiologist at the Memorial Sloan-Kettering Cancer Center in New York City, there are approximately 1 million new cases of nonmelanoma skin cancer annually with about 1,200 deaths, and about 40,000 new melanoma cases with 7,200 deaths. Melanoma metastasizes readily, while the nonmelanomas rarely do.

According to session organizer Francis Gasparro, director of the Jefferson University Photobiology Laboratory at Thomas Jefferson University in Philadelphia, 21 FDA-approved compounds are potentially available for use in sunscreens marketed in the United States. However, most of the research into their photochemistry has been done by industry, and the results are not available to either the public or academic researchers. Many sunscreens protect against some part of the ultraviolet (UV) spectrum, Gasparro says, but none of the sunscreens available perform "like a layer of concrete on your skin."

Hoping to unravel the connections between melanoma, long-wave ultraviolet radiation (UVA), and short-wave ultraviolet radiation (UVB), biophysicist Richard Setlow of the Brookhaven National Laboratory in New York exposed light-sensitive tropical fish to UVA. He found a high incidence of melanoma induction. Setlow suggests that if the fish results are transferable to humans, sunscreens formulated to block only UVB do not offer reasonable protection against melanoma.

Two studies reported at the February meeting sought to determine whether sunscreen protects DNA from UV damage. In a study funded by a consortium of pharmaceutical and cosmetics manufacturers, Honnavara Ananthaswamy, professor and deputy chairman of the department of immunology at the University of Texas M.D. Anderson Cancer Center in Houston, and his team tracked the rate of mutation in the *p53* tumor suppressor gene in mice exposed to UVB. Results of the study, published in the May 1997 issue of *Nature Medicine*, showed that after 16 weeks, in mice pretreated with sunscreen with a sun protection factor (SPF) of 15, *p53* mutations were almost nonexistent, Ananthaswamy reports, whereas 50% of the mice without sunscreen showed the mutation after 12 weeks. All of the mice without sunscreen developed skin tumors after 41 weeks of daily exposure. None of the mice treated with sunscreen developed skin tumors during this time or even after 54 weeks of continuous sunscreen and UV exposure. Ananthaswamy says the *p53* mutation can serve as a very early warning of nonmelanoma skin cancer induction.

He would like to see what he calls an "MPF" (mutation protection factor) added to the SPF designation on sunscreens.

John Knowland, a researcher in the department of biochemistry at Oxford University in the United Kingdom, studied whether sunscreen compounds become chemically reactive in the presence of UV light and pass their excess energy to DNA. In his study, published in the August 1997 issue of *Photochemistry and Photobiology*, Knowland exposed both naked DNA and cultured human cells treated with padimate O, a derivative of para-aminobenzoic acid, to UV radiation in the laboratory. In both cases he observed DNA strand breakage, presumably caused by hydroxyl radicals.

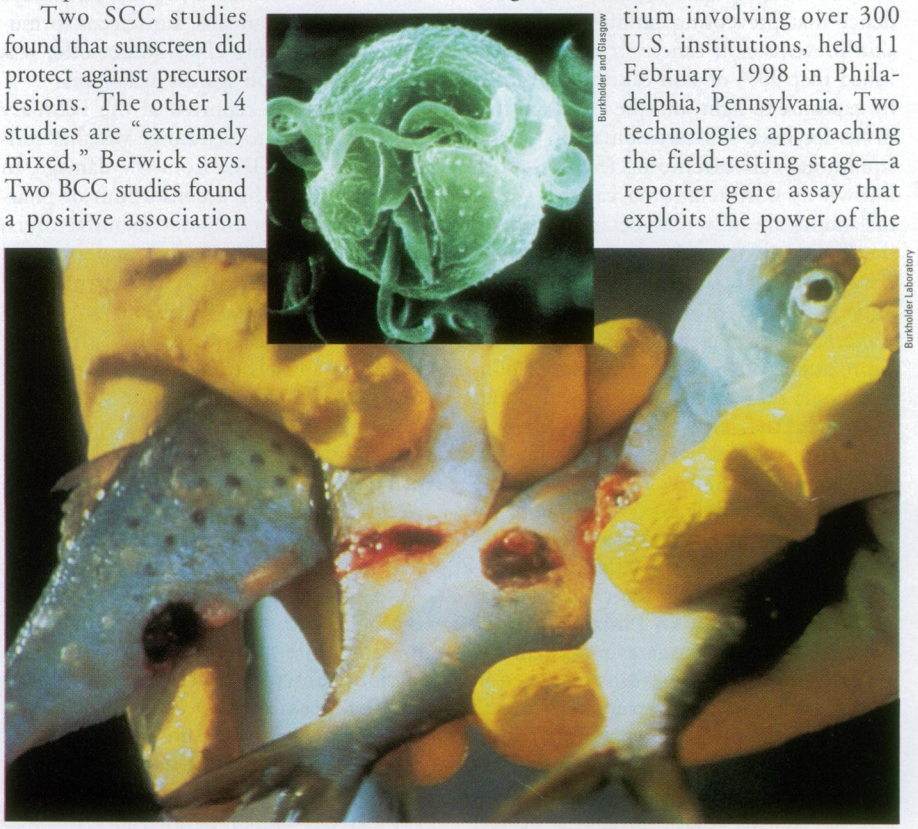
Thus, one suppressor gene study shows promise for sunscreens in the prevention of nonmelanoma skin cancers, and the other shows that at least one sunscreen component itself actually induces DNA damage. The epidemiological evidence is equally confusing. Berwick surveyed 16 epidemiological studies, and says that these studies show that "squamous cell carcinoma is associated with continuous sun exposure, basal cell carcinoma seems to be associated with continuous [and] intermittent sun exposure on the unadapted skin, and melanoma seems to be associated with intermittent, intense sun exposure on untanned, unadapted skin."

Two SCC studies found that sunscreen did protect against precursor lesions. The other 14 studies are "extremely mixed," Berwick says. Two BCC studies found a positive association between the use of sunscreen and the incidence of BCC. Of 10 melanoma studies, five showed a positive association between the use of sunscreens and the development of melanoma. Two showed sunscreen to be protective, and three showed no association.

"We can conclude from these studies that it is not safe to rely on sunscreen to protect you from getting skin cancer," Berwick says. She notes that the positive association between sunscreen and melanoma may be due to the possibility that for people at highest risk for developing melanoma (light-skinned, light-eyed people, especially those with many moles), sunscreen may bestow a false sense of security. They may stay out in the sun longer than they would otherwise. Berwick emphasizes that until more clarifying research is done, people should pay close attention to their skin-cancer risk factors and reduce their sun exposure accordingly.

Researchers Ready Rapid *Pfiesteria* Tests

By 1999 or sooner, field tests to rapidly identify *Pfiesteria piscicida* and its toxins—responsible for massive fish kills and reports of serious human health effects—could help prevent illness, researchers hinted during a briefing of the National Sea Grant College Program, a research and education consortium involving over 300 U.S. institutions, held 11 February 1998 in Philadelphia, Pennsylvania. Two technologies approaching the field-testing stage—a reporter gene assay that exploits the power of the firefly's glow-making gene, and DNA-based molecular probes—were described in greater detail during a subsequent February 18–19 scientific conference in Linthicum, Maryland. At the same time, policy makers, environmental officials, and industrial leaders continue to discuss options for reducing pollutants that might play a role in the emergence of *Pfiesteria*.



Testing for toxins. Researchers are developing rapid tests for toxins from *Pfiesteria* zoospores that cause lesions and death in fish.

A single-celled dinoflagellate—the so-called "cell from hell"—*Pfiesteria* literally flagellates or whips through water in some of its forms, and its toxins paralyze fish, allowing the organism to feed on their tissues. Public fears concerning *Pfiesteria* escalated last year after Maryland epidemiologists reported profound learning disabilities and short-term memory loss sustained by some people who were exposed to the organism, according to JoAnn Burkholder, an associate professor of botany at North Carolina State University in Raleigh, who was one of the discoverers of the organism.

Though the chemical structure of *Pfiesteria* toxins remains a mystery, boatside and blood tests are now under development by John S. Ramsdell, a branch chief for the National Oceanographic and Atmospheric Administration's (NOAA) Marine Biotoxins Program in Charleston, South Carolina, and Parke Rublee, an associate professor of biology at the University of North Carolina at Greensboro. Both men are collaborating with Burkholder.

In Miami, Florida, meanwhile, researchers at the NIEHS Marine and Fresh Water Center at the University of Miami are scrambling to purify and characterize several different *Pfiesteria* toxins, says Daniel G. Baden, the facility director. With J. Glenn Morris, a researcher at both the University of Maryland's School of Medicine and that university's Center for Marine Biotechnology, the Miami-based NIEHS group also is studying toxin-related physiological and neuropsychological changes in humans and sheep, Baden reports.

To develop a detection system, Ramsdell's research group spliced together the section of a firefly gene that codes for the glow-producing enzyme luciferase with part of a human gene, *c-fos*, which is sensitive to *Pfiesteria* toxins. The resulting reporter gene was then inserted into a rat cell that also responds to the toxic algae. The result, Ramsdell says, is a genetically engineered cell capable of giving off light when it comes in contact with the targeted toxins.

Light can then be measured on a luminometer. "The amount of light emitted by the cell is proportional to the amount of